OIPE JC154
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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant:	**Keith D. Allen *et al.***	Group Art Unit:	**1632**
Serial No.:	**10,015,948**	Examiner:	**Ton, Thaian N.**
Filed:	**December 11, 2001**	Customer No.	**26619**
Title:	**Transgenic Mice Containing Adrenocorticotropin Hormone Receptor Gene Disruptions**	Docket/Order No.	**R-605**
		Date:	**July 28, 2003**

RESPONSE TO RESTRICTION REQUIREMENT

Commissioner for Patents
Mail Stop Non-Fee Amendments
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

In response to the Office Action mailed May 27, 2003 concerning the Examiner's restriction to the claims in connection with the above-referenced application, Applicants elect without traverse Group II (claims 3-9 and 14-28). Applicants submit concurrently herewith a Petition for an Extension of Time under 37 CFR § 1.136(a) for response to the Office Action for a period of two (2) months from May 27, 2003 up to and including July 28, 2003, with July 27, 2003 falling on a Sunday.

Respectfully submitted,

Date: 7/28/03

Kelly L. Quast, Reg. No. 52,141
Deltagen, Inc.
700 Bay Road
Redwood City, CA 94063
(650) 569-5100

CERTIFICATE OF MAILING UNDER 37 CFR 1.8

I hereby certify that this correspondence and its listed enclosures is being deposited with the United States Postal Service as First Class Mail, postage paid, in an envelope addressed to: Commissioner for Patents, Alexandria, VA, Mail Stop Non- Fee Amendment/OIPE on **July 28, 2003**.

Name: **Don Mixon**

Signed: Don Mixon Date: 7/28/03

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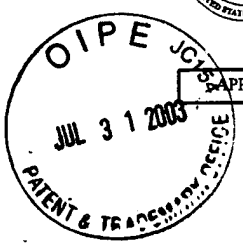


APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/015,948	12/11/2001	Keith D. Allen	R-605	2942

7590 05/27/2003

DELTAGEN, INC.
740 Bay Road
Redwood City, CA 94063

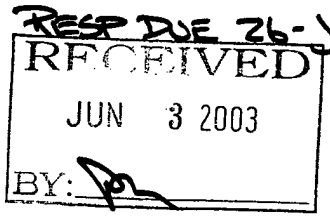
EXAMINER
TON, THAIAN N

ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 05/27/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

RESP DUE 26-JUNE-03



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Office Action Summary

Application No.	Applicant(s)
10/015,948	ALLEN ET AL.
Examiner	**Art Unit**
Thai-An N. Ton	1632

-- *The MAILING DATE of this communication appears on the cover sheet with the correspondence address* --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 1 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____ .

2a)☐ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) 1-38 is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) _____ is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☒ Claim(s) 1-38 are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

Claims 1-38 are pending.

Election/Restrictions

Restriction to one of the following inventions is required under 35 U.S.C. 121:

I. Claims 1-2, drawn to a targeting construct and a method of producing the gene-targeting construct, classified in class 536, subclass 23.1.

II. Claims 3-9, 14-28 drawn to a genetically modified non-human animal comprising a disruption in a ACTHR gene, cells, methods of using the cells to producing a genetically modified non-human animal, a non-human transgenic animal and transgenic mice comprising a disruption in a ACTHR gene, classified in class 800, subclass 3, 8, 21, 25; class 435, subclass 455, 463, 320.1, 325.

III. Claim 10, drawn to methods of identifying an agent that modulates the function of a ACTHR gene in vivo, by determination of whether the function of a disrupted ACTHR gene is modulated, classified in class 800, subclass 3.

IV. Claims 11-12, drawn to methods of identifying an agent that modulates the expression of a ACTHR gene in vitro, classified in class 435, subclass 4, 6.

V. Claim 13, drawn to an agent, unclassifiable.

VI. Claim 29, drawn to methods of identifying an agent that ameliorates a phenotype associated with a disruption in a HIP1 gene using a transgenic mouse comprising a disruption in a HIP1 gene, classified in class 800, subclass 3.

VII. Claim 30, drawn to an agent, unclassifiable.

VIII. Claim 31, drawn to methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR, unclassifiable.

IX. Claims 32, drawn to methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR, unclassifiable.

X. Claim 33, drawn to a pharmaceutical composition, classified in class 530, subclass 350+, for example.

XI. Claim 34, drawn to methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse, classified in class 800, subclass 3.

XII. Claim 35, drawn to methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse classified in class 800, subclass 3.

XIII. Claim 36, drawn to a method of identifying an agent that inhibits the activity or function of ACTHR, *in vitro*, classified in class 435, subclass 4.

XIV. Claim 37, drawn to an agonist or antagonist of ACTHR, unclassifiable.

XV. Claim 38, drawn to phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene, classified in class 702, subclass 19.

The inventions are distinct, each from the other because of the following reasons:

Inventions I and II are distinct. The nucleic acid construct can be used as probe while the cells can be used in *in vitro* assays. The transgenic non-human animal of Invention I can be used to observe HIP1 gene function or as a model for disease or condition.

Inventions I and any of Inventions III-XV are mutually exclusive and independent. The nucleic acid construct of Invention I is not required for the implementation of methods of identifying an agent that modulates the function of a

ACTHR gene in vivo, by determination of whether the function of a disrupted ACTHR gene is modulated of Invention III, the methods of identifying an agent that modulates the expression of a ACTHR gene in vitro of Invention IV, the agent of Invention V, the methods of identifying an agent that ameliorates a phenotype associated with a disruption in a ACTHR gene using a transgenic mouse comprising a disruption in a ACTHR gene of Invention VI, the agent of Invention VII, the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Inventions II and any of Inventions III, IV, VI, XI, XII and XV are related as product and process of use. The inventions can be shown to be distinct if either or both of the following can be shown: (1) the process for using the product as claimed can be practiced with another materially different product or (2) the product as

claimed can be used in a materially different process of using that product (MPEP § 806.05(h)). In the instant case the transgenic non-human animal or transgenic mice of Invention II can be used as a model for disease or condition and the cells of Invention II can be used to produce ACTHR protein *in vitro*.

Invention II and any of Inventions V, VII-X, XIII and XIV are mutually exclusive and independent. The transgenic non-human animals of Invention II are not required for the agent of Invention V, the agent of Invention VII, the methods of treating susceptibility to seizure of Invention VIII, the methods of treating hyperactivity of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that inhibits the activity or function of ACTHR of Invention XIII, and the agonist or antagonist of Invention XIV, and vice versa.

Invention III and any of Inventions IV-XV are mutually exclusive and independent. The methods of identifying an agent that modulates the function of a ACTHR gene in vivo, by determination of whether the function of a disrupted ACTHR gene is modulated of Invention III is not required for the implementation of the methods of identifying an agent that modulates the expression of a ACTHR gene in vitro of Invention IV, the agent of Invention V, the methods of identifying an agent that ameliorates a phenotype associated with a disruption in a ACTHR gene using a transgenic mouse comprising a disruption in a ACTHR gene of Invention VI, the agent of Invention VII, the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of

ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa. Furthermore, each of the methods requires a separate and materially different protocol.

Invention IV and any of Inventions V-XV are mutually exclusive and independent. The methods of identifying an agent that modulates the expression of a ACTHR gene in vitro of Invention IV are not required for the implementation of the methods of identifying an agent that ameliorates a phenotype associated with a disruption in a ACTHR gene using a transgenic mouse comprising a disruption in a ACTHR gene of Invention VI, the agent of Invention VII, the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of

identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa. Furthermore, each of the methods requires a separate and materially different protocol.

Invention IV and Inventions V are distinct because the agent of Invention V can be identified different methods and the methods of Invention IV are not required for the agent.

Invention V and any of Inventions VI-XV are mutually exclusive and independent. The agent of Invention V is not required for the implementation of the methods of identifying an agent that ameliorates a phenotype associated with a disruption in a ACTHR gene using a transgenic mouse comprising a disruption in a ACTHR gene of Invention VI, the agent of Invention VII, the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of

identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention VI and any of Inventions VIII-XV are mutually exclusive and independent. The methods of identifying an agent that ameliorates a phenotype associated with a disruption in a ACTHR gene using a transgenic mouse comprising a disruption in a ACTHR gene of Invention VI are not required for the implementation of the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a

transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention VI and Inventions VII are distinct because the agent of Invention VII can be identified different methods and the methods of Invention VI are not required for the agent.

Invention VII and any of Inventions VIII-XV are mutually exclusive and independent. The agent of Invention VII is not required for the implementation of the methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII, the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention VIII and any of Inventions IX-XV are mutually exclusive and independent. The methods of treating susceptibility to seizure comprising to a subject in need a therapeutically effective amount of ACTHR of Invention VIII are

not required for the implementation of the methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX, the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention IX any of Inventions X-XV are mutually exclusive and independent. The methods of treating hyperactivity comprising to a subject in need a therapeutically effective amount of ACTHR of Invention IX are not required for the pharmaceutical composition of Invention X, the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention X and any of Inventions XI-XV are mutually exclusive and independent. The pharmaceutical composition of Invention X are not required for the implementation of the methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI, the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention XI and any of Inventions XII-XV are mutually exclusive and independent. The methods of identifying an agent that ameliorates susceptibility to seizure by administration of the agent to a transgenic mouse of Invention XI are not required for the methods of identifying an agent that ameliorates hyperactivity by administration of the agent to a transgenic mouse of Invention XII, the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention XII and any of Inventions XIII-XV are mutually exclusive and independent. The methods of identifying an agent that ameliorates hyperactivity

by administration of the agent to a transgenic mouse of Invention XII are not required for the implementation of the method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII, the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Invention XIII and either of Inventions XIV or XV are mutually exclusive and independent. The method of identifying an agent that inhibits the activity or function of ACTHR *in vitro* of Invention XIII is not required for the agonist or antagonist of Invention XIV, and the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Inventions XIV and XV are mutually exclusive and independent. The agonist or antagonist of Invention XIV is not required for the phenotypic data associated with a transgenic mouse comprising a disruption in a ACTHR gene of Invention XV, and vice versa.

Because these inventions are distinct for the reasons given above and have acquired a separate status in the art as shown by their different classification and their recognized divergent subject matter and because the searches for the groups are not coextensive, restriction for examination purposes as indicated is proper.

Applicant is advised that the reply to this requirement to be complete must include an election of the invention to be examined even though the requirement be traversed (37 CFR 1.143).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Thái-An N. Ton whose telephone number is (703) 305-1019. The examiner can normally be reached on Monday through Friday from 8:00 to 5:00 (Eastern Standard Time), with alternating Fridays off. Should the examiner be unavailable, inquiries should be directed to Deborah Reynolds, Supervisory Primary Examiner of Art Unit 1632, at (703) 305-4051. Any administrative or procedural questions should be directed to William Phillips, Patent Analyst, at (703) 305-3482. Papers related to this application may be submitted to Group 1600 by facsimile transmission. Papers should be faxed to Group 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center number is (703) 872-9306.

TNT

Thái-An N. Ton
Patent Examiner
Group 1632

DEBORAH CROUCH
PRIMARY EXAMINER
GROUP ~~1800~~ 1630